Three decades of research have outlined serious consequences of anabolic androgenic steroid (AAS) use on the haemostatic system. Recent work has shown raised levels of C reactive protein (CRP) to be a strong predictor of cardiovascular events. CRP concentrations have not been studied in AAS users to date.

MATERIALS AND METHODS
Ethical approval for the study was granted by the Bro Taf local health authority. Subjects were divided into three groups: AAS users who were still using AAS at time of testing (n = 10); bodybuilding controls who did not use any pharmacological ergogenic aids (n = 10); sedentary male controls (n = 8). Venous blood was sampled, from which serum concentrations of C reactive protein, male sex hormones, and cardiac troponin T were determined.

RESULTS: A significantly altered hormonal profile in the AAS using group provided indirect confirmation of AAS use. C reactive protein concentrations were significantly (p<0.05) higher in the AAS using bodybuilders. There was no relation between C reactive protein and cardiac troponin T.

Conclusion: AAS using bodybuilders had significantly higher C reactive protein concentrations, indicating a greater propensity to develop peripheral arterial disease.

<table>
<thead>
<tr>
<th>Variable</th>
<th>AAS users (n = 10)</th>
<th>Bodybuilding controls (n = 10)</th>
<th>Sedentary controls (n = 8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Testosterone (nmol/l)</td>
<td>41 (26.1)*</td>
<td>17 (3.7)</td>
<td>15 (3.00)</td>
</tr>
<tr>
<td>SHBG (nmol/l)</td>
<td>4.0 (2.8)**</td>
<td>13 (8.4)</td>
<td>21 (11.1)</td>
</tr>
<tr>
<td>Free androgen index</td>
<td>10.3***</td>
<td>1.3</td>
<td>0.7</td>
</tr>
<tr>
<td>CRP (mmol/l)</td>
<td>1.2 (0.5)*</td>
<td>0.7 (0.3)</td>
<td>0.5 (0.2)</td>
</tr>
<tr>
<td>Troponin T (mumol/l)</td>
<td>&lt;0.1</td>
<td>&lt;0.1</td>
<td>&lt;0.1</td>
</tr>
</tbody>
</table>

Values are mean (SD). *p<0.05; **p<0.01; ***p<0.001 compared with both controls.

SHBG, Sex hormone binding globulin.

DISCUSSION
The mechanism for AAS induced CRP alterations is not known. An absence of a concurrent increase in troponin T in the AAS using group indicates inflammation at a source other than the myocardium. CRP is secreted by hepatocytes in response to in vivo inflammatory events. Indeed, much biological activity of AAS also centres around the liver. This possible link certainly warrants more detailed investigation.

This study adds to the list of potentially prothrombotic consequences of non-therapeutic AAS use, and provides a contraindication to such use.

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Abbreviations: AAS, anabolic androgenic steroid; CRP, C reactive protein
REFERENCES

Data supplements
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